

ALTERNOBARIC VERTIGO

AN EXPERIMENTAL STUDY IN MAN OF VERTIGO
DUE TO ATMOSPHERIC PRESSURE CHANGES

by

ÖRJAN TJERNSTRÖM

Malmö 1974

Hund

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The studies on which this thesis was based were supported by the Delegation for Applied Medical Defence Research (Project Nos. U 65/1972, U 87/1972 and U 95/1973), the Swedish Medical Research Council (No. B 73-17X-99-09) and by Malmö General Hospital.



Printed in offset.

Malmö General Hospital

Malmö 1974.

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I. Introduction, the student, A. B. C. of the subject, p. 101.

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TO MY KID(S)

The present thesis is based on the following papers:

- I. Ingelstedt, S., Ivarsson, A. & Tjernström, Ö. 1974.
Vertigo due to relative overpressure in the middle ear -
an experimental study in man. Acta Otolaryng (Stockh.),
Preprint.
- II. Tjernström, Ö. 1974. Middle ear mechanics and Alternobaric
Vertigo. Acta Otolaryng (Stockh.), Preprint.
- III. Tjernström, Ö. 1974. Further studies on Alternobaric
Vertigo - posture and passive equilibration of middle ear
pressure. Acta Otolaryng (Stockh.), Preprint.
- IV. Tjernström, Ö. 1974. Alternobaric Vertigo in divers -
an experimental study. Undersea Biomedical Research.
Accepted for publication.

In the text these papers will be referred to by their Roman
numerals.

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INTRODUCTION

Vertigo following exposure to changes in atmospheric pressure has been known for a long time and was first recognized in divers and caisson workers (e.g. Curnow, 1894; Alt, Heller, Mager & von Schrötter, 1897; Keays, 1909). Most of the episodes were interpreted as due to haemorrhages in the labyrinths or due to decompression sickness with bubble formation in the inner ears, and in some of the cases the pressure changes apparently had a traumatic effect and caused long-lasting damage or permanent lesions in the inner ears.

There is, however, a more functional type of vertigo caused by atmospheric pressure changes and due to non-traumatic degrees of inadequate pressure equilibration of the middle ears. This type of vertigo, which is usually of short duration, was recognized as early as 1927 (Peyser) and discussed 1937 by Armstrong & Heim. It seems, however, that little account has been taken of this phenomenon in modern literature on aviation and diving medicine. Yet over the last two decades attention has been focused on this transient type of vertigo, which has been encountered in both flyers (Melvill Jones, 1957; Armstrong, 1961; Lundgren & Malm, 1966; Enders & Rodriguez-Lopez, 1970; Brown, 1971) and divers (Taylor, 1959; Coles & Knight, 1961; Lundgren, 1965; Vorosmarti & Bradley, 1970) and has become known as Alternobaric Vertigo (A.V.) (Lundgren, 1965). The opinion of several authors is that A.V. is probably more common than has been generally realized. Thus Melvill Jones (1957) reported an incidence of 10 % in military pilots and Lundgren & Malm (1966) found that 17 % of 108 military pilots flying modern jets had experienced A.V. Melvill Jones also predicted an increase in the incidence of A.V. with the performance of modern military aircrafts as a result of the very rapid changes in altitude.

The frequency of experience of A.V. among divers has been demonstrated by, among others, Lundgren (1965) and Vorosmarti & Bradley (1970), who

found that 26 % out of 354 sports divers and 12 % out of 143 military divers had experience of A.V.

The importance of A.V. as a source of danger is highlighted by case reports illustrating the difficulties pilots as well as divers might encounter. Thus Melvill Jones (1957) reported how a young fighter pilot during rapid ascent "felt his ears suddenly clear, and at the same instant experienced complete, although transient, disorientation. All points of visual reference, both inside and outside the cockpit, had the appearance of rotating. He found it impossible to refer to his instruments. The situation was so precarious that he was on the point of escaping by means of his ejection seat when everything settled down and he was able to regain formation." Case reports dealing with divers with experience of A.V. often describe disorientation and difficulties to find the way to the surface. Thus one diver in Lundgren's study reported as follows: "I started the ascent. When I was only about one metre above the bottom vertigo started and increased, finally becoming unbearable when I was only a few metres up. I got immediate relief on returning to the bottom. Two more attempts to ascend ended in the same way. Finally I got hold of my friend's waist and was pulled to the surface. During the ascent the vertigo was very intense. I had no idea of what was up and what was down - it was impossible to fix any objects visually."

In a recent interview study (Lundgren, Tjernström & Örnhausen, 1974) answered by 2053 Swedish divers, 343 were considered presumably to have had A.V., as no other cause than changes in ambient pressure seemed likely. Out of these divers about 50 % had experienced difficulties in orientation, 20 % had suffered nausea and an equal proportion had had to abort dives during which A.V. was encountered. The importance of A.V. as a danger in diving should be related to the increasing interest in sports diving, as illustrated by the Swedish Sport Divers Association with 550 members in 1965, 4000 in 1971 and 7000 members in 1973.

There is a surprising lack of knowledge of the pathophysiologic mechanism in the cases where the disturbance of the inner ear function is of a transient nature. As for the mechanism of A.V., anamnestic information obtained from divers and flyers has given some clues. Most authors agree that the vertigo is caused by a relative over-pressure in the middle ears, since most subjects who have some experience of A.V. have observed that it occurs when the middle ear pressure has been increased indirectly by reduction of the ambient pressure at ascents or directly by blowing against a clamped nose (Valsalva's manoeuvre). Some authors, however, are of the opinion that even an ambient pressure increase might cause vertigo (e.g. Armstrong & Heim, 1937; Loch, 1942; Haines & Harris, 1946; DeWeese & Saunders, 1960), and it has also been suggested that the vertigo is produced by abrupt changes of pressure within the middle ears (e.g. Melvill Jones, 1957; Hilger, 1959; Armstrong, 1961; Benson, 1965). As for the mechanism by which pressure changes may induce vertigo in normal subjects, it has been proposed that the pressure somehow induces an unusually large displacement of the stapes footplate and thus sets the inner ear fluid in motion, thereby stimulating the vestibular receptors (e.g. Hilger, 1959; Fields, 1958; Heller, 1960). Some authors consider the mechanism by which vertigo is induced akin to the "fistula-test" mechanism. Thus Melvill Jones (1957) and Benson (1965) suggest that the bony wall separating the middle ear cavity from the horizontal canal may be so thin in certain subjects as to become deformed when the middle ear pressure differs from the ambient pressure. The relatively gradual pressure build-up during ascent or descent would thus cause a moderate fluid displacement too slow and insufficient to deflect the cupula. However, when a sudden change in the pressure takes place, as when the middle ear is cleared, any displacements which may have occurred will be suddenly reversed and the sudden fluid flow may be expected to generate "eddy currents" stimulating the vestibular receptors. However, as pointed out by Lundgren (1965), vertigo under field conditions appears frequently to last too long (several minutes) to be caused by sudden movements of the stapes (cf. Vorosmarti & Bradley,

1970; Lundgren et al., 1974).

Thus the mechanism by which atmospheric pressure changes affect the vestibular system is unknown. The explanations given have been based on anatomical and physiological considerations as well as on anamnestic information obtained from subjects with experience of A.V. An experimental approach to the phenomenon has so far been lacking.

The main purpose of the present studies was to investigate if it might be possible to elicit A.V. in the laboratory; to find out, by means of electronystagmography, if the sensation of vertigo is of vestibular origin; to study if the pressure changes required for a vestibular stimulation are of a kind that might be encountered during flying and diving; to study the mechanism by which pressure changes affect the vestibular system; to find out if in certain subjects individual qualities predispose to this type of vertigo; to investigate if divers who have experienced A.V. during diving might differ from divers without such experience with regard to the tubal function, and finally if it might be possible to eliminate "vertigo-prone" subjects applying for flying and diving service.

I. VERTIGO DUE TO RELATIVE OVERPRESSURE IN THE MIDDLE EAR - AN EXPERIMENTAL STUDY IN MAN

Reduction of ambient pressure induces relative overpressure in the middle ear, and if the tube is not opened actively the relative overpressure gradually increases with the ambient pressure reduction so that there will finally be a passive opening of the tube. Most episodes of A.V. have been observed during ascents and the vertigo has been suggested to be due to a relative overpressure in the middle ears. It seems conceivable that there might be a relation between the occurrence of A.V. and the relative overpressure in the middle ears induced during ascents, since most pilots and divers usually do not clear their ears actively at ascents.

The aim of the present study was to determine the level of relative overpressure in the middle ears that might result from ambient pressure decrease, i.e. the level of overpressure necessary to force the Eustachian tubes open (forcing pressure level - FPL); to find out if such overpressure can cause A.V. in normal subjects; and to investigate, by means of electronystagmography, if experimentally elicited A.V. might be of a vestibular origin.

Method and procedure

The subject was encased in a pressure chamber in which it was possible to simulate ascents and descents by pressure changes amounting to 90 cm H₂O in 25 sec. According to Ingelstedt, Ivarsson & Jonson (1967) and Elner, Ingelstedt & Ivarsson (1971_a) pressure changes in the middle ears, induced by ambient pressure changes, were studied indirectly at physiological conditions (intact eardrums). The following recordings were performed: volume displacement of the eardrum (V_{tm}) and chamber pressure (P_{ch}). (The volume displacement of the eardrum was induced by pressure changes in the chamber.) Simultaneously with the recordings of V_{tm} and P_{ch}, recordings of eye-movements by means of electronystagmography (ENG) were performed. An outline of the equipment used in the

experiments is given in Fig. 1.

The subjects were examined in the sitting position in total darkness, instructed to keep their eyes open and avoid blinking and exposed to simulated ascents and descents both with and without active clearing of their ears. The sudden opening of the Eustachian tube, due to the relative overpressure in the middle ear induced by reduction of ambient pressure, appeared clearly on the Vtm-curve (Fig. 2). Hence it was possible to derive, from the Pch-curve, the ΔP_{ch} required to cause a passive opening of the tube.

Because of the elastic properties of the structures of the middle ear, including the eardrum, the actual relative overpressure in the closed middle ear, i.e. the pressure gradient across the eardrum (P_{tm}), must be somewhat lower than the ΔP_{ch} . Calculations have to be performed for a correct determination of the P_{tm} necessary to force the tube open passively, and knowledge is required of the middle ear pressure (P_m) as well as of the ambient pressure (P_{ch}) at the moment of passive opening. Before each test the eardrum was checked for a neutral position, which means that $P_{ch} = P_m$, i.e. $P_{tm} = 0$. Fig. 3 provides an outline of the different variables involved. At the moment of passive forcing, the pressure gradient across the eardrum (P_{tm}) is equal to the difference between the pressure change in the chamber (ΔP_{ch}) and the pressure change in the middle ear (ΔP_m), i.e. $P_{tm} = \Delta P_{ch} - \Delta P_m$. Determination of the middle ear pressure change (ΔP_m) during changing ambient pressure (ΔP_{ch}) is possible by using Boyle's law according to Ingelstedt et al. (1967) (see equation 2). This calculation requires knowledge of the middle ear volume (V_m), which in the present study had to be determined indirectly by means of a roentgenological - planimetric method (Diamant, 1940), by which the area of the air-filled middle ear system was measured from X-ray films and then converted to volume according to Andréasson (1973). The middle ear volume has to be corrected for the volume variation of the mucosa lining the tympanic cavity. This volume variation, caused by pressure changes in the middle ear, has been studied (Andréasson, Ingelstedt, Ivarsson, Jonson & Tjernström, 1974) and determined at $0.5 \mu\text{l/cm H}_2\text{O}$ in the

experimental conditions used in the actual investigations.

Results

The passive opening of the Eustachian tubes occurred at different pressure levels in different subjects, and the distribution of 79 subjects with regard to the passive opening of the tubes in relation to the pressure decrease in the chamber is given in Fig. 4. The mean ΔP_{ch} required to force the tubes open was determined at 43.9 cm H₂O. Reexaminations of 14 subjects at different sessions and with intervals of several months showed a high reproducibility of the individual FPL values (Table I). 5 subjects reported vertigo when exposed to simulated ascents and passive clearing of the ears, 2 of them (indicated in Fig. 4 by black squares) reported vertigo at every test and the other 3 only occasionally. Horizontal nystagmus recorded simultaneous with the vertigo episodes indicated that the vertigo was of a vestibular origin. Vertical nystagmus was never seen. These 5 subjects were found to have a particularly high FPL on one side ($\Delta P_{ch} > 60$ cm H₂O). FPL asymmetry between the left and the right ear was always observed. However, the importance of the pressure asymmetry with regard to inner ear stimulation could not be evaluated.

Fig. 5 demonstrates that the vestibular stimulation was not induced at the moment of pressure regulation but during the period of asymmetric middle ear pressure, and not until the pressure in the not-cleared ear had reached a certain level. Calculations of the P_{tm} showed only a relatively small difference between P_{tm} and ΔP_{ch} (Tables IV and VI). This was due to the fact that in all subjects examined for the middle ear volume (V_m) a normal or a large V_m was found (> 6 ml, Tables III and V), and it is seen from equation 2 that in the case of a small V_m the difference is greater.

It was also of interest to calculate the P_{tm} and the pressure asymmetry between the two ears at the moment nystagmus appeared (nystagmus pressure level - NPL). As is seen from Table VII also the individual NPL seems to be quite constant from one session to the other. The elicited nystagmus was always found to be directed towards the ear

which had the highest FPL. No nystagmus was seen during ascents with active equilibration and no vertigo was reported. Descents with and without active equilibration caused neither vertigo nor nystagmus. The 5 subjects with a high FPL could all, by means of deglutitions, actively equilibrate static as well as dynamic over- and underpressures in a normal way and belonged to tubal function groups Ib and Ic (very good tubal function) according to the tubal function groups presented by Elner et al. (1971_b).

Conclusion

Certain subjects, although otologically normal, are more susceptible to A.V. because of an inclination to a permanent high FPL on one side. The demonstrated vestibular stimulation was not induced at the moment of pressure regulation as an effect of sudden movements of the stapes, but during the period of asymmetric middle ear pressure and not until the relative overpressure in the "not-cleared" ear had reached a certain level. The importance of the magnitude of the pressure asymmetry between the two ears could not be evaluated. There was no relation between the active equilibration capacity and the FPL. A relative underpressure in the middle ears, within a range of 90 cm H₂O, could not be shown to affect the vestibular system. Experiments with frequent equilibration also during ascents, performed with the vertigo-prone subjects, demonstrated that it might be possible to prevent A.V., since no vestibular stimulation was recorded during these tests.

The mechanism by which a relative overpressure in the middle ear affects the vestibular system is still unknown. The lateral displacement of the eardrum might cause the stapes footplate to move against the pressure gradient due to the mechanical linkage to the eardrum, and a simultaneous displacement of the round window membrane, so that it bulges into the inner ear, might conceivably cause an inner ear fluid displacement sufficient to stimulate the vestibular system mechanically.

II. MIDDLE EAR MECHANICS AND ALTERNOBARIC VERTIGO

In view of the results presented in paper I, it seemed of interest to study the way in which the functional components of the middle ear (eardrum and the ossicular chain) might be responsible for the inner ear stimulation recorded at ambient pressure changes. The main purpose was to assess the importance for the inner ear stimulation of the outward displacement of the eardrum, caused by the relative overpressure in the middle ear at simulated ascents. Another aim was to prove the assumption that the vestibular stimulation recorded at simulated ascents (paper I) was elicited from the ear with the highest FPL.

Two otologically healthy subjects, who had reported experience of A.V. at every single test when exposed to simulated ascents and passive clearing of the ears in the laboratory, were selected for the present study.

Method and procedure

Recordings of V_{tm} and P_{ch} as well as calculations of P_{tm} were performed as described in paper I.

By means of the equipment shown in Fig. 3, over- and underpressure was induced locally in the external ear canal.

Recordings of eye-movements by means of ENG were performed simultaneously with the different procedures.

The inner ear stimulation demonstrated during simulated ascents and passive clearing of the ears (paper I) was assumed to be elicited from the middle ear with the highest FPL, which was found to be the right ear of both subjects of the present study.

Four different procedures were used:

- (1) Simulated ascents and passive clearing of both ears.
- (2) Underpressure induced locally in the right external ear canal in order to cause an outward displacement of the eardrum. The underpressure was controlled so as to cause an outward displacement of the eardrum of the same degree as that during simulated ascents

and passive clearing of the ears.

Before the next step of the investigations the right eardrum was perforated and a transmyringal plastic tube was inserted into the eardrum to offer free air communication between the middle ear and the external ear canal.

(3) Simulated ascents and passive clearing of the left ear without inducing overpressure in the right ear owing to the transmyringal tube.

(4) Overpressure induced locally in the right external ear canal. Owing to the transmyringal tube the pressure in the middle ear followed the pressure in the external ear canal. The speed of the pressure change was controlled so as to be the same as that recorded at the passive forcing of the tubes with intact eardrums. The pressure increase in the external ear canal was interrupted before the Eustachian tube was forced open passively in order to avoid airflow through the middle ear, which might otherwise cause caloric stimulation.

Results

Simulated ascents and passive clearing of both ears elicited vertigo as well as nystagmus in both subjects (Fig. 4). The outward displacement of the eardrum in the experiments with underpressure in the external ear canal (intact eardrum) caused no sign of vestibular stimulation. Simulated ascents and passive clearing of only the left ear induced roughly the same asymmetry (Fig. 5) as that recorded in the experiments with passive forcing of both Eustachian tubes (cf. Fig. 4), and despite the same asymmetry no vertigo appeared. In the experiments at which an overpressure was induced locally into the middle ear via the transmyringal tube, it was possible to elicit nystagmus of about the same intensity and starting at about the same pressure level (Fig. 5) as was seen in the experiments with passive clearing of both ears (cf. Fig. 4).

Conclusion

The prerequisite for a vestibular stimulation caused by overpressure in the middle ear seems to be that the overpressure should be of a certain level in relation to the ambient pressure. Asymmetry in middle ear pressure is not sufficient *per se* for vestibular stimulation (not even in vertigo-prone subjects). Movements of the eardrum *per se* could not be shown to have any effect on the vestibular system. It was also observed that approximately the same vestibular stimulation was elicited under the two different experimental conditions with overpressure in the middle ears, although the eardrum was immobilized by a transmyringal tube in one of the situations. The possibility that compression or expansion of the middle ear gas volume might cause temperature changes inducing caloric stimulation was also discussed and excluded.

III. FURTHER STUDIES ON ALTERNOBARIC VERTIGO -

POSTURE AND PASSIVE EQUILIBRATION OF MIDDLE EAR PRESSURE

It is well known that the pressure-equalizing ability of the Eustachian tubes is affected by different postures (Lucae, 1867; Hartman, 1879; Perlman, 1939) and the reduced tubal patency seen in the recumbent position has been assumed to be caused by venous engorgement of the tubal mucosa corresponding to the vascular filling of the middle ear mucosa (Ingelstedt et al., 1967; Rundcrantz, 1969).

The present study consists of two parts. In part I, 5 otologically healthy subjects, who were known to have a forcing pressure (FPL) of a normal level ($\Delta P_{ch} < 50 \text{ cm H}_2\text{O}$) in the sitting position, were examined. In part II, 6 otologically healthy subjects, who were known to have a high FPL on one side ($\Delta P_{ch} > 60 \text{ cm H}_2\text{O}$) in the sitting position, were examined.

The aim of part I was to investigate the influence of different body positions on the passive equilibration of the middle ears on normal subjects. The aim of part II was to investigate, in view of the results of part I, the possibility of increasing even a high FPL by change of posture. The aim of part II was also to investigate, in view of the demonstrated relation between a high FPL and the occurrence of A.V. paper I, if a higher relative overpressure in the middle ear might cause a stronger vestibular response.

Method and procedure

Recordings of Vtm and Pch as well as calculations of Ptm were performed as described in paper I. In part I the FPL was expressed as ΔPch . In part II the FPL was expressed as Ptm, and the relative overpressure recorded at the moment nystagmus started - nystagmus pressure level (NPL) - was also calculated.

Recordings of eye-movements by means of ENG were performed simultaneously with the recordings of Vtm and Pch.

The subjects were examined in the sitting and the recumbent position. In some experiments also the left and right lateral position was used. Experiments were also performed with the subjects in the recumbent position, with simultaneous neck vein compression, by means of an inflatable cuff (Jonson & Rundcrantz, 1969).

Results (part I)

The FPL was found to be higher in the recumbent position than in the sitting position and the mean increase was about 13 cm H₂O (mean of all means). 3 subjects were also examined in the left and the right lateral position and the recordings showed a higher FPL in the ear which was turned downwards than in the ear turned upwards (Table I). No sign of vestibular stimulation was observed.

Results (part II)

The results (Table II) demonstrated that an initially high FPL

(recorded in the sitting position) did not increase as regularly and not to the same extent as an initially low FPL (cf. Table I).

Neck vein compression caused an additional FPL increase.

All subjects but one (subject VII, Table II) reported vertigo when examined in the sitting position, and nystagmus was also recorded. One subject reported vertigo in both positions (sitting and recumbent), but in the other 4 subjects, who had reported vertigo in the sitting position, no sign of vestibular stimulation was observed in the recumbent position in spite of the fact that in this position the recorded FPL was apparently high enough to stimulate the vestibular system in the sitting position.

In view of these results the relative overpressure required to cause a vestibular stimulation in the recumbent position seemed to be higher than that in the sitting position. Actually, when the relative overpressure recorded at the moment nystagmus appeared (nystagmus pressure level - NPL) was calculated, it was found that this level was higher in the recumbent than in the sitting position. As appears from Table IV, the mean NPL was always found to be higher (5-8 cm H₂O) in the recumbent position and an additional increase (5-9 cm H₂O) was furthermore seen in the examinations during neck vein compression. The mean NPL was quite constant from one session to another as long as the examinations were performed in the same position, despite varying levels of the FPL (Tables II and III). The graphical presentation in Figs. 3, 4 and 5 seems to indicate a stronger vestibular response at higher FPL. However, the findings are not statistically conclusive. Furthermore, Figs. 3 and 4 also show that the magnitude of the pressure asymmetry between the two ears of the same individual did not seem to affect the intensity of the vestibular response.

Conclusion

The pressure level required to force the tubes open was higher in the supine than in the sitting position. The assumption that the postural effects on the tubal patency is that of vascular filling of the tubal mucosa due to increased hydrostatic venous pressure in the recumbent

position (Jonson & Rundcrantz, 1969) was further confirmed by the experiments performed with neck vein compression, which caused an additional FPL increase.

It was demonstrated in paper II that approximately the same vestibular response was elicited whether the overpressure in the middle ear was induced indirectly by ambient pressure reduction or directly by inducing the overpressure locally in the middle ear. Thus the pre-requisite for vestibular stimulation seemed to be a certain relation between the middle ear pressure and the ambient pressure. However, the results of the present study indicate that in actual fact the pre-requisite might be a certain relation between the inner and the middle ear pressure.

IV. ALTERNOBARIC VERTIGO IN DIVERS - AN EXPERIMENTAL STUDY

Information about A.V. in divers has so far been available only in epidemiologic studies (Taylor, 1959; Coles & Knight, 1961; Lundgren, 1965; Terry & Dennison, 1966; Bayliss, 1968; Vorosmarti & Bradley, 1970). However, information received in interview studies could only to a limited extent allow conclusions about the nature of vertigo and a better foundation for such conclusions would be obtained if the phenomenon could be studied under laboratory conditions.

As for the mechanism of A.V. anamnestic information obtained from divers has provided some clues. Most divers with experience of vertigo during diving have observed that vertigo starts during ascents. The suggestion that relative overpressure in the middle ears is important for the development of A.V. has gained further support from some divers observation that the vertigo may be relieved by interrupting the ascent and descending again (Lundgren, 1965).

A.V. was earlier investigated in an experimental study of non-diving subjects (papers I, II and III) and a relation was found between a

unilateral high FPL and the occurrence of vertigo during simulated ascents. In a recent epidemiologic study of A.V. on a large group of divers (Lundgren, Tjernström & Örnhausen, 1974) questions concerning asymmetry in passive equilibration during ascents were also asked. (It was explained to the divers that a sensation of air passage through the Eustachian tubes is a symptom of asymmetry.) The answers to these questions revealed that such asymmetry was experienced by significantly more divers with, than by divers without, experience of A.V. ($p < 0.001$).

In view of the observations in the non-diving material (papers I and III) it was considered interesting to apply the same method to divers in order to elucidate possible differences in active as well as passive equilibration capacity between subjects who had experienced A.V. during diving and subjects who had not.

The subjects were selected from the above-mentioned interview study (Lundgren et al., 1974) which was in part undertaken with the aim of recruiting vertigo-prone subjects for the present experimental study.

Method and procedure

Recordings of Vtm and Pch were performed as described in paper I. The FPL was given as ΔPch .

Recordings of eye-movements were performed simultaneously with the recordings of Vtm and Pch.

The active equilibration capacity was investigated by applying positive and negative Pch-changes and using the Vtm-curve (Fig. 2) as a criterion of equilibration according to Elner et al. (1971_b).

The subjects were exposed to simulated ascents and passive clearing of their ears in the sitting and the recumbent position. The latter position was tried because it is known to influence the FPL (paper III), presumably by causing venous engorgement in the mucosa of the Eustachian tube (Ingelstedt et al., 1967; Rundcrantz, 1969). It was also thought to offer an approximate parallel to the circulatory changes which might be induced by immersion in water (e.g. Arborelius, Balldin, Lilja & Lundgren, 1972).

Results

The results are presented in Table I. 12 divers with experience of A.V. during diving were found to have a significantly higher mean FPL than 6 divers without history during diving ($p < 0.001$). Furthermore, 6 out of the 12 divers with a high FPL reported vertigo in the laboratory and nystagmus recorded simultaneously with the vertigo episodes indicated that the latter was of a vestibular origin. Shifting to the recumbent position always caused an increase in the FPL. However, this postural change did not induce vertigo in subjects who had not reported vertigo in the sitting position. One possible explanation of this might be the fact that the overpressure required for vestibular stimulation also increases in the recumbent position (paper III). When active equilibration of middle ear pressure was performed during the ascents there were no episodes of vertigo or nystagmus.

Conclusion

The results confirm the connection between a high FPL and the occurrence of vertigo which was earlier shown in a non-diving material (papers I and III). It is also obvious that it might be possible to eliminate subjects susceptible to A.V. by investigations of the passive equilibration during simulated ascents, since in view of the present results it is conceivable that subjects who experience A.V. during diving could to a great extent be expected to belong to a group of non-diving population which was shown to have a high FPL and to be susceptible to A.V. in the laboratory (paper I). It is also demonstrated that it might be possible to prevent A.V. by active equilibration also during ascents, since it prevents inducement of high overpressure in the middle ear.

In the above-mentioned interview study (Lundgren et al., 1974) divers with experience of vertigo during diving reported greater difficulties in achieving pressure equilibration than divers without such experience. The results of the present study showed a statistically significant

difference between the two groups with regard to the passive equilibration during ascents, but not with regard to the active equilibration capacity. However, the conditions under which the active equilibration was tested differ greatly from those which a diver is faced with during actual diving. Therefore a difference in active pressure equilibration capacity which makes itself felt during actual diving might not necessarily become obvious under the less strenuous conditions in the laboratory. Neither the present experimental studies nor those of Elner et al. (1971_b) or those presented in paper I demonstrated any connection between the active tubal function and the magnitude of the recorded FPL.

GENERAL DISCUSSION

The investigations presented in this thesis have demonstrated that vertigo, as a symptom of inner ear stimulation, might occur as a result of only moderate ambient pressure changes and following relative overpressure in the middle ear built up by reduction of ambient pressure. This type of inner ear stimulation should be regarded as a functional reaction to pressure changes in the middle ear since there were no signs of traumatic lesions to the inner ear structures. Most of the literature on vertigo due to ambient pressure changes deals with vertigo as a symptom of inner ear damage causing long-lasting or permanent lesions in the inner ear (e.g. Curnow, 1894; Alt, 1896; Boot, 1913; Freeman & Edmonds, 1972; Edmonds, 1973) or as a symptom of decompression sickness (e.g. Alt et al., 1897; Keays, 1909; Erdman, 1913; Vail, 1929; Bühlmann & Waldvogel, 1966; Farmer, 1973; Lång & Rózsahegyi, 1973). Very little account has been taken of the more physiological type of vertigo.

Relative overpressure in the middle ear seems to be crucial for eliciting A.V. However, there are reports on transient vertigo also during descents, and some authors are of the opinion that even a relative underpressure in the middle ears might cause vertigo (Armstrong & Heim, 1937; Loch, 1942; Haines & Harris, 1946; De Weese & Saunders, 1960). Yet, in the present study, it was not possible to record any vestibular stimulation during experiments with underpressure (within the range of 0 - 90 cm H₂O) in the middle ears. Actually, a number of the transient vertigo episodes observed during descents have been reported together with some tubal dysfunction, and the vertigo was observed concurrently with Valsalva's manoeuvre (Lundgren & Malm, 1966; Enders & Rodriguez-Lopez, 1970; Brown, 1971) - thus a relative overpressure might even in these cases have been responsible for the vestibular stimulation.

Simulated ascents and passive clearing of the ears were thought to offer an approximate parallel to conditions that may be encountered in actual flying and diving, since most pilots and divers usually do not clear their ears actively during ascents but wait for the ears to clear passively. The passive equilibration was found to occur at different pressure levels in different subjects and even at different levels between the two ears of the same individual. The subjects who reported vertigo during ascents were found to have a particularly high FPL on one side and repeated examinations, with intervals of several months, demonstrated that the individual variation of the FPL from one session to another was low. Thus it appears that some subjects, although otherwise otologically normal, are more likely to experience A.V. because of an inclination to a permanently high FPL on one side. Why in some individuals a higher middle ear pressure is required to force open the Eustachian tube could not be explained in the present study.

Some of the most frequent causes for reduced tubal patency are acute or chronic infections of the upper respiratory tract and inflammatory conditions of the Eustachian tubes or middle ears. As a matter of fact, a great many A.V. episodes have been reported concurrently with early stages or resolutions of common colds (Melvill Jones, 1957; Benson, 1965; Lundgren & Malm, 1966; Vorosmarti & Bradley, 1970; Enders & Rodriguez-Lopez, 1970; Brown, 1971) and it has been suggested that pathological processes affecting either the middle ear or the upper respiratory tract are prerequisites for the occurrence of A.V. The subjects of the present investigations were, however, otologically normal, and it was always checked that they were free from signs of catarrhal infections prior to the experiments. Thus, other factors than those mentioned above must have been responsible for the high FPL values and further studies are in preparation in order to evaluate the nature of these factors.

Since it was early recognized that ascents rarely caused discomfort in the ears, interest has mainly been focused on the function of the Eustachian tube during descents. The mechanism of the active opening

of the tube has been studied and analyzed by several authors (e.g. Zöllner, 1942; Perlman, 1951; Aschan, 1955; Flisberg, Ingelstedt & Örtengren, 1963; Ingelstedt et al., 1967; Rundcrantz, 1969) but only few studies have paid attention to the factors determining the pressure level required to force the tubes open passively (e.g. Hartman, 1879; Armstrong & Heim, 1937; Flisberg et al., 1963). The experiments in the present study performed with the subjects in the recumbent position, which demonstrated a FPL increase with change of posture, might give some clues. The recumbent position is known to cause a venous engorgement of the middle ear mucosa and it has been suggested that a corresponding congestion of the tubal mucosa might be responsible for the reduced tubal patency observed in the recumbent position (Perlman, 1939; Ingelstedt et al., 1967; Rundcrantz, 1969). This assumption was further confirmed by the present experiments with neck vein compression which caused an additional FPL increase.

Conceivably the mechanical factors, operating the closure of the Eustachian tube, might just as well affect the passive opening. Thus the pressure exerted by the tissues surrounding the tube (Zöllner, 1942; Guild, 1955) as well as the spring effect of the tubal cartilage might be relevant. The elastic properties of the tissues supporting the membranous tube (e.g. Zöllner, 1942; Aschan, 1955; Guild, 1955) might be more or less important, and it is conceivable that a more rigid structure requires a higher FPL. In the case of a more rigid structure of the tissues supporting the tube, it might be expected that the extraluminal hydrostatic pressure would have a smaller effect than in the case of more elastic structures surrounding the tube. This reasoning might give a possible explanation of the finding that an initially high FPL increased less with change to the recumbent posture than an initially low FPL. The surface tension of the tubal mucus might be another factor of importance to the passive opening of the tube. Flisberg et al. (1963) found that if saline was used in the middle ear to force open the tube, a smaller overpressure (5 mm Hg) was required than if air was used (20 mm Hg) and this was attributed to a reduction in surface tension.

When discussing the patency of the Eustachian tube it should be noted that there was no relation between the active and the passive tubal equilibration capacity. Most of the subjects who had a permanent high FPL could actively equilibrate overpressure as well as underpressure in a normal way. However, the magnitude and rate of pressure changes in actual flying and diving greatly exceed those applied in the present laboratory studies, and especially divers run the risk of barotrauma which might cause oedema of the tubal mucous membrane. Melvill Jones (1957) also suggested that "the current practice of breathing 100 % oxygen under all conditions of flight" might increase the incidence of A.V. as there is evidence to indicate that a high partial pressure of oxygen might have an irritating effect upon the mucous membranes of the nasopharynx and the middle ear. Such an oxygen effect was demonstrated by Aschan (1948). Immersion in water induces circulatory changes (e.g. Arborelius et al., 1972) and the examinations of the present study with the subjects in the recumbent position were in part performed in order to provide an approximate parallel to such circulatory changes.

Actually, some preliminary experiments (3 subjects) have been performed (Ivarsson, Lundgren, Tjernström & Örnhammar, 1974) in which the subjects were exposed to simulated ascents during immersion in water (sitting with the head above the water surface). The FPL did increase due to immersion, though less (about 50%) than when the subject assumed the recumbent position (dry).

Thus several factors are involved in actual flying and diving which conceivably contribute to a reduced tubal patency.

Although much is known about the labyrinthine function in man, there is a lack of knowledge of the mechanism by which non-traumatic degrees of pressure changes in the middle ear affect the inner ear and induce A.V. Several pilots and divers have reported that vertigo was preceded by a clicking sound, as when air leaves or enters the middle ear cavity. Since most of the explanations of the A.V. mechanism have so far been based on anamnestic information received from subjects with experience

of A.V., it has been assumed by several authors that A.V. is produced by abrupt changes of pressure as when the ears are cleared (Melvill Jones, 1957; Hilger, 1959; Armstrong, 1961; Benson, 1965; Enders & Rodriguez-Lopez, 1970). Probably the most accepted explanation as to how such sudden pressure changes affect the inner ear is that the pressure changes are transferred through the oval window by movements of the stapes by means of the mechanical linkage of the eardrum and the ossicular chain (e.g. Melvill Jones, 1957; Fields, 1958). Furthermore, "unusual oval and round window anatomy" (Hilger, 1959) as well as a thin bony wall separating the horizontal canal from the middle ear cavity (Melvill Jones, 1957; Benson, 1965) have been suggested as prerequisites. Yet it was seen from the present studies that the vestibular stimulation was not induced by abrupt changes in pressure, as for instance as an effect of sudden movements of the stapes, but during a relatively slow ambient pressure reduction ($4 \text{ cm H}_2\text{O/sec}$). It was also observed that the vestibular response (nystagmus) started before the subjects observed vertigo. In many experiments the sensation of vertigo was felt to start near the moment at which the ear was cleared passively, and this might give an explanation of the above-mentioned assumption that A.V. is elicited by means of sudden pressure changes in the middle ear. The vestibular stimulation was induced during a period of asymmetric middle ear pressure and this asymmetry was due to the different FPL of the right and the left ear. It appears conceivable that any asymmetrical stimulation of the vestibular system would alter the balanced inputs from the resting discharge of the vestibular receptors and thus cause sensations of vertigo and other appropriate responses such as nystagmus. However, in the present study it was demonstrated that no vestibular stimulation was recorded until the overpressure in the middle ear had reached a certain level (stimulation threshold - NPL). Thus asymmetric middle ear pressure *per se* is not sufficient for eliciting a vestibular response, not even in "vertigo-prone" subjects, as demonstrated in the experiments with perforation of one eardrum. The demonstration of the NPL also explained the finding that varying degrees of asymmetric pressure between the two ears, recorded at

different examinations, did not influence the intensity of the vestibular response.

Vorosmarti & Bradley (1970) suggested that subjects who had experienced A.V. perhaps might be more sensitive to pressure changes in the middle ears than others. Yet it would seem to appear from the present studies that the prerequisite is a relative overpressure in the middle ears high enough to reach the stimulation threshold. Although the results indicated some individual variation as to the required pressure level, no vestibular response below a relative overpressure of about 55 cm H₂O was ever observed. Unilateral vestibular hyporeflexia has also been suggested as an explanation of an asymmetric stimulation of the vestibular system caused by pressure changes in the middle ears during ascents, even under symmetrical pressure conditions in the middle ear cavities (Edmonds et al., 1973). In view of the present investigations, however, not even such a vestibular hypofunction might be responsible for A.V. unless the FPL is sufficiently high.

It was also observed that the effect of overpressure in the middle ear was roughly the same, whether or not there was a simultaneous lateral displacement of the eardrum. Actually, the middle ear structures seem to protect the inner ear from large amplitudes of eardrum displacement (Békésy, 1960). Békésy demonstrated that in the case of large eardrum displacements the stapes rotated around an axis running longitudinally through the footplate; "then the fluid of the inner ear merely shifts from the upper half of the footplate to the lower without causing any displacement". Thus the overpressure seems to affect the vestibular system "directly" and not by way of movements of the stapes as an effect of eardrum movements. In order to evaluate the importance of pressure transfer to the inner ear via the oval window, preliminary experiments (2 cases) of simulated ascents have been performed during which the stapedius muscle was kept contracted by means of a tone stimulus of 115 dB (1000Hz) in the other ear. This, however, could not be shown to change the stimulation caused by the relative overpressure in the middle ear.

As to the mechanism by which middle ear pressure changes affect the intralabyrinthine pressure, Bezold (1908) demonstrated that the rise or fall in the latter mainly depended upon the mobility of the round window membrane. However, the mechanism by which the middle ear overpressure affects the vestibular system is still unknown, and possible factors are inner ear fluid displacement as well as blood circulation disturbance. Further studies which pay attention to these factors are being prepared.

The direction of the nystagmus seems to indicate a "stimulation", i.e. an increased discharge of impulses from the vestibular receptors of the affected ear. Such an activation might reflect a genuine stimulation following for example an utriculopetal cupula deviation in the horizontal semicircular canal, but might alternatively be due to a disturbance (hypoxia) of the neurophysiological mechanism responsible for the discharge of vestibular impulses (pseudo-stimulation).

In view of the discussion above, the work of Nylén & Karlefors (1921) has to be mentioned. These authors could show that a constant overpressure in the middle ear, in subjects with an eardrum perforation, caused nystagmus which gradually increased in strength during 10 - 20 seconds and then ceased. In their opinion the mechanism is an "impression on the yielding portions of the labyrinth, which would cause a lymphatic movement with accompanying irritation". It should, however, also be mentioned that these authors performed their experiments on subjects suffering from various ear diseases, and pathological conditions might thus have been contributory factors.

The demonstrated NPL increase when the subject assumed the recumbent posture was an interesting finding since little is known about factors which influence the intralabyrinthine pressure.

The increase in NPL might reflect an increase in the intralabyrinthine pressure. The intracranial pressure is known to increase by about 10 - 13 cm H₂O when the posture is changed from the erect to the

horizontal position (Best & Taylor, 1945). If the cochlear and/or the endolymphatic ducts have a pressure-equalizing function (Allen, 1964; House, 1964; Holden & Schuknecht, 1968) the intralabyrinthine pressure would be expected to rise when the intracranial pressure is increased. Alternatively, since the increase of the intracranial pressure is mainly an effect of a hydrostatic venous pressure increase (Dawson, 1967), it might be assumed that the intralabyrinthine pressure would rise as a result of the same mechanism even without any pressure-equilibrating function on the part of the ducts.

It is the opinion of the present author that A.V. constitutes an important potential danger to pilots and divers. Most episodes are, however, of short duration and not very troublesome, which might explain why such episodes are spontaneously reported only to a limited extent. It may also explain the opinion of some authors that A.V. is a rare and insignificant phenomenon. The dissimulation of a pilot afraid of losing his flying license might also prevent episodes of A.V. from becoming known. There are, however, case reports illustrating that A.V. might cause real trouble and it is possible that the most serious incidents are never revealed at all.

Practical implications and recommendations

It appears from the present study that some subjects are more susceptible to A.V. than others and that it might be possible to eliminate such subjects when applying for diving and flying service by examining the passive equilibration capacity of the middle ears. It appears obvious that it is not sufficient to test the active tubal function only, as no relation between the active and the passive equilibration ability was found. If the present method will be used for routine examinations of otologically healthy applicants, it seems sufficient to determine only the ΔP_{ch} required to force the Eustachian tubes open, since only small differences between the P_{tm} and the ΔP_{ch} in subjects with normal ears ($V_m > 6$ ml) were demonstrated. Besides a good selection of diving and flying personnel as well as careful

operational practice with respect to upper respiratory infections, information about the Eustachian tube function seems to be important. Many divers with experience of A.V. have pointed out that active equilibration - also during ascents - eliminates or greatly reduces the risk of A.V. According to Coles (1973), knowledge and teaching of Eustachian tube physiology is supposed to have contributed to the very low incidence of "Alternobaric accidents" in the British Royal Navy.

With the very rapid increase in sports diving it seems difficult to apply the above-mentioned method of selection on a major scale. However, applicants for professional diving occupation should undergo the test. Furthermore, in the selection of applicants for flying modern high performance aircrafts the method should be recommended. While a diver with knowledge of the mechanism of A.V. might conceivably cope with a temporary disorientation, a sudden onset of such disorientation might be fatal to a pilot. Actually, Melvill Jones in 1957 indicated the future need for a method devised with a two-fold object (1) "of providing a means of investigating the matter further" and (2) "of providing something in the nature of a clinical test which could be applied to aircrew suspected of having an unusual susceptibility".

Not only sports diving but "sports flying" is growing rapidly. Many applicants, as well as subjects renewing their flying license, are today refused owing to various kinds of previous ear diseases, since it may be difficult to assess their reaction to ambient pressure changes. By applying the present method, some of these subjects might possibly satisfy the requirements.

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